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Summary

Bile acids in the gut-liver axis

Kai Markus Schneider

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Digestive Oncology | Immunology | Hepatobiliary

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The speaker presented bile acids as dynamic signaling molecules shaped by gut microbiota that play critical roles in cholestatic liver disease, with microbial bile acid metabolism essential for maintaining the enterohepatic circulation and potential implications for stratifying FXR-targeted therapies in PSC.

KEY POINTS

- In a mouse model of cholestatic liver disease, depletion of the gut microbiome led to accumulation of conjugated bile acids, loss of intestinal FXR activation, bile infarct formation, and severe weight loss requiring experiment termination after seven days, a phenotype rescued by FXR agonist treatment.
- The speaker reported that in PSC patients, suppressed bile acid synthesis measured by the surrogate marker C4 was associated with poor outcome and performed almost as well as the Mayo risk score for prognosis.
- Patients with suppressed bile acid synthesis showed an increased ratio of primary to secondary bile acids, and the PRIME trial testing Cilofexor for PSC was stopped for early futility, though a subgroup with earlier disease showed benefit.
- The speaker stated that approximately 95 percent of bile acids are reabsorbed in the terminal ileum, with the majority of secondary bile acid metabolism occurring in the colon where microbial enzymes diversify the bile acid pool.
- The speaker presented data showing that loss of microbial bile acid metabolism disrupts the FGF15-mediated negative feedback loop suppressing hepatic bile acid synthesis, leading to toxic accumulation in cholestatic conditions.
- The speaker suggested that FXR-targeted therapies in cholestatic disease should be stratified by FXR activity, as bile acid synthesis is already suppressed in many advanced PSC patients.

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